Distribution of Blood Lead Levels in a Birth Cohort of New Zealanders at Age 21

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Little is known about lead exposure in the general population of young adults. In this study, whole blood lead concentration (PbB) was determined in a sample of the Dunedin Multidisciplinary Health and Development Study, a well-documented birth cohort of New Zealanders aged 21 years in 1993–1994. PbB in those who consented to venipuncture at 21 years of age (n = 779; 411 males, 368 females) was compared to PbB for the same cohort at age 11 years. The PbB at age 21 ranged from 0.4 to 56 µg/dl with a geometric mean of 4.5 µg/dl (95% CI, 4.3–4.7 µg/dl). Only three individuals had a PbB above 30 µg/dl. Males had significantly higher PbB than females (geometric mean 6.0 vs. 3.2 µg/dl; $p<0.0001$). The PbB at age 21 was 53% lower than in the same individuals at age 11 (geometric mean 4.8 vs. 10.2 µg/dl; $p<0.001$; n = 480) and the correlation between corresponding values was weak (r = 0.19; $p<0.001$). PbB at age 21 showed significant associations with high risk occupational activities, recreational exposure, domicile close to a main road, smoking, and male sex. Blood lead concentrations continue to fall in New Zealand, but occupational and recreational activities remain a significant source of lead exposure. *Key words*: birth cohort, blood lead concentration, lead exposure, longitudinal study, New Zealand, risk factors. *Environ Health Perspect* 104:1332–1335 (1996)

Environmental lead contamination is widespread in industrialized societies and poses a number of health hazards (*1*). In general, children are much more at risk from lead exposure than adults due to higher rates of absorption of ingested lead (*2*) and its ability to impair cognitive development (*3,4*). The effects of chronic low-level exposure in adults have not been defined, but cognitive deficits have been detected in populations with only moderate occupational exposure (*5,6*) and a more pervasive effect on systolic blood pressure occurs in the general population (*7*).

Monitoring for lead exposure generally involves measuring the concentration of whole blood lead (PbB). In addition to effects on cognitive functioning, adverse biochemical and hematological effects of lead can be detected at a PbB as low as 10 µg/dl (*8*). Mean blood lead levels well in excess of this value have been found in a number of cross-sectional blood lead studies carried out in samples of children from the general population (*9*). Even a rural/urban sample of 11-year-old New Zealand children participating in the Dunedin Multidisciplinary Health and Development Study (DMHDS) had a geometric mean PbB of 10.3 µg/dl when studied in 1983–1984 (*10,11*).

Occupational exposure limits have become more stringent over recent years. In New Zealand, the Occupational Safety and Health Service of the Department of Labour now recommends that workers with a sustained PbB of 54 µg/dl or above

should be suspended (*12*). The effects of low-level lead exposure are the subject of intense research at present, but in the absence of detailed knowledge, occupational hygiene measures including worker education and personal protective equipment remain important, particularly in high risk occupations (*13*).

Efforts to control lead exposure require accurate information on PbB levels in specific populations. In the United States, PbB levels have shown a substantial decline in the entire population, probably due to the elimination of leaded gasoline in 1986 (*14*). However, higher blood lead levels continue to be associated with younger age, male sex, non-Hispanic black race, and low income level. In New Zealand, roughly half of lead absorption in the urban population has been shown to arise from leaded gasoline (*15*), but the removal of this additive has followed a much slower course than in the United States and its elimination was not complete until early 1996. The consequences of this require ongoing surveillance of PbB levels in the general New Zealand population.

Little information is available concerning lead levels in populations of young adults. The DMHDS involves a birth cohort of approximately 1000 individuals with well-documented personal and occupational life histories who were recently studied at 21 years of age. The aims of this study were to determine the PbB in the DMHDS sample at age 21 (1993–1994), to compare this with PbB at age 11

(1983–1984), and to examine PbB in relation to three general categories of exposure risk: residential, occupational, and recreational.

Methods

Subjects. The DMHDS is a longitudinal study based on all children born at Queen Mary Hospital, Dunedin, New Zealand, between 1 April 1972 and 31 March 1973 of mothers residing in Dunedin (*16*). Dunedin is a city of approximately 120,000 inhabitants situated close to the southern tip of New Zealand's South Island in the Province of Otago. Of 1,139 children resident in the Otago region at the time of their third birthdays, consent was given for 1,037 to participate in the study. The cohort was followed up every second year from age 3 to 15 and again at 18 and 21 years of age. The number of participants at each phase of the study has been consistently high (>90%) and the cohort is representative of young New Zealanders except in its ethnic mix, which is largely Caucasian with fewer Maori/Polynesians (about 3%) than the country as a whole (about 12%). Of the total sample of the DMHDS surviving at 21 years of age (n = 1,020; 526 males, 494 females), a total of 949 (484 males, 465 females) attended on a day close to their twenty-first birthdays and completed the lead exposure questionnaire. A further 40 members were contacted by telephone. Of those seen, 779 sample members (411 males, 368 females) consented to venipuncture for the lead study. The study was approved by the Southern Regional Health Authority Ethics Committee (Otago), and informed consent was obtained for blood sampling.

Assays. At about 4:00 P.M. on the assessment day, venous blood samples (7 ml) were collected into lithium heparin (Vacutainer) tubes and stored at -20°C until assayed. Blood taken into separate

tubes containing tripotassium EDTA as anticoagulant was used for measurement of packed cell volume (PCV) and hemoglobin concentration. PbB was determined after a fivefold dilution of blood in 0.2% Triton X-100 on a Varian AA 40 atomic absorption spectrometer (Varian Australia, Mulgrave, Victoria) using a graphite furnace and Zeeman background correction (*17*). PbB is reported in micrograms per deciliter (1 µg/dl = 0.0483 µmol/l). The assay has a limit of detection of 0.4 µg/dl and an average coefficient of variation of less than 5%. Quality control of the lead assay was monitored by analysis of three samples monthly in the Quality Assurance Program of Standards Australia. Performance by the laboratory in this program showed the slope and intercept were not significantly different from one and zero, respectively, with a reproducibility of 0.93 µg/dl.

Lead exposure. Exposure to lead from residential, occupational, and recreational factors 'was assessed by questionnaire. With regard to dwelling, members were asked whether they lived in the country, a small town, a small city, a suburb of a large city, or near a large city center; if they lived within 50 meters of a busy road (>12 vehicles/min at peak traffic); and if they lived in a brick, roughcast, wooden, or other type of house. With regard to occupation, they were asked whether, as part of their occupation, they had been involved in any of the following high risk activities (*18*) during the last year: radiator repair, car mechanical work, engine reconditioning, muffler repair, panel beating, metalwork/welding, battery repair, scrap-metal work, foundry work, boat building, or house painting. The high risk category included those who were involved in one or more of these activities. For each individual activity, subjects were also asked to indicate whether respiratory protection was used. With regard to recreational activities, they were asked whether they had never, rarely, sometimes, or often been involved in any of the following lead-related leisure activities during the past year: metalwork/welding; soldering; working on cars; making lead-lights, bullets, or sinkers; or small-bore rifle shooting. The high risk category included those who were often involved in one or more of these activities. Information on smoking and alcohol use was available from other parallel studies. To obtain smoking frequency, the participants were asked whether they usually smoked on a daily basis and the number of cigarettes smoked. They were divided into nonsmokers, those smoking <10 cigarettes per day, and those smoking ≥10 cigarettes per day. To assess alcohol use, they were asked how many days per month they drank
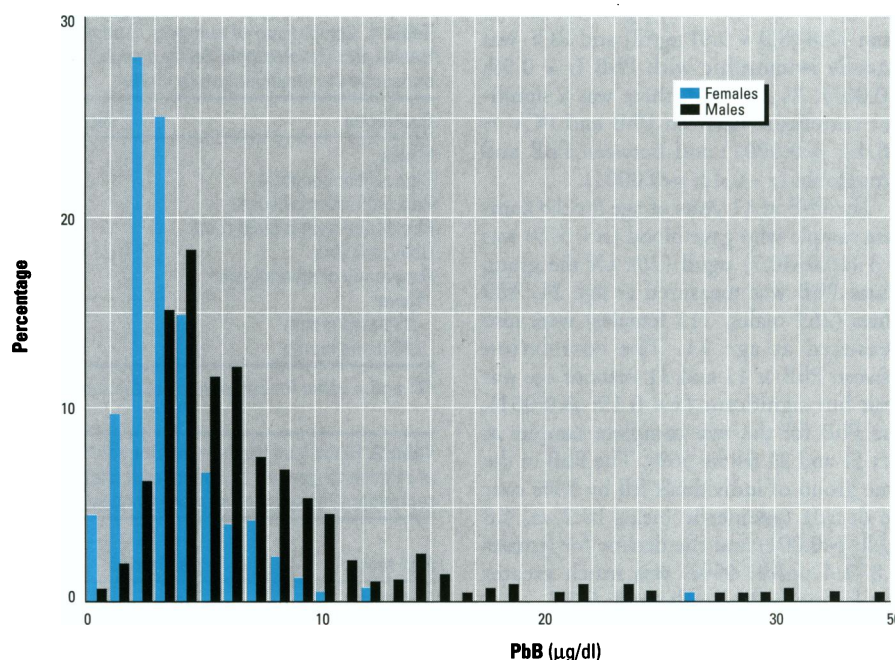

Figure 1. Distribution of whole blood lead concentration (PbB) for 411 males and 368 females participating in the Dunedin Multidisciplinary Health and Development Study at 21 years of age.

alcoholic beverages and were divided into those who never drank, those drinking on less than 15 days/month, and those drinking on 15 or more days/month. Socioeconomic status (SES) of the sample members' parents for the sample at age 15 were available based on the 1976 Elley–Irving scale, which uses education, achievement, and income (*19*). For the purposes of this study, those with either a father or mother in one of the categories 1–4 (professional or skilled workers) were designated as high SES and compared with those in categories 5 and 6 designated as low SES.

Statistical methods. Red cell lead (RCL) was calculated by dividing the PbB by the PCV. Data were analyzed using SPSS, the Software Package for Social Sciences (SPSS Inc., Chicago, IL). The distribution plot of PbB (Fig. 1) was positively skewed (skewness = 3.83) so a logarithmic transformation was used to normalize the PbB distribution (skewness = -0.17) for parametric analysis. Data for PbB and RCL are given as geometric mean [95% CI] and arithmetic mean (standard deviation; SD). Multiple regression analysis was used to evaluate the association between log transformed PbB and the various risk factors. A value of $p<0.05$ was taken as statistically significant.

Results

The place of residence of the group giving blood was given as Dunedin (65.8%), the province of Otago (5.5%), elsewhere in New Zealand (21.6%), and overseas

(7.1%, mainly in Australia). A comparison of those giving blood with the rest of the DMHDS cohort (i.e., those not seen or not giving blood) showed those giving blood had significantly higher qualifications upon leaving school but were not significantly different for gender or the SES of their families at age 15. For those who completed the lead exposure questionnaire, a comparison of lead exposure between the group giving blood and the group who did not consent to give blood is shown in Table 1. The group giving blood contained proportionally more males and fewer daily smokers, but in terms of residence near a main road, high risk occupation, recreational activity, or use of alcohol, they were no different from the group declining to give blood.

The distribution of PbB is shown in Figure 1 for both males and females. The distributions were highly skewed approaching log-normal distributions. The range of PbB was from 0.4 to 56 µg/dl with a geometric mean of 4.5 (95% CI, 4.3–4.7) µg/dl and an arithmetic mean of 5.6 (SD = 4.6) µg/dl. Forty-seven individuals (5 females) had PbB values above 10 µg/dl, 27 (1 female) had PbB values above 15 µg/dl, and only 3 males had PbB greater than 30 µg/dl. The geometric means for males and females were 6.0 (95% CI, 5.7–6.4) and 3.2 (95% CI, 3.1–3.4) µg/dl, respectively [arithmetic means 7.2 (SD = 5.4) and 3.8 (SD = 2.3) µg/dl, respectively], and this difference was statistically significant ($p<0.0001$). The geometric mean RCL was

10.6 (95% CI, 9.9–11.0) µg/dl [arithmetic mean 12.8 (SD = 9.9) µg/dl] and RCL was virtually isomorphic with PbB (r = 0.99; $p<0.001$). As expected, there was a significant correlation between PbB and PCV (r = 0.42; $p<0.0001$) and between PbB and hemoglobin (r = 0.42; $p<0.0001$).

The PbB at 11 years of age for the complete sample who gave blood (n = 579) was 10.3 (10.0–10.7) µg/dl (*10*). Of the group whose PbB was measured at age 21, 480 values (269 males, 211 females) were also measured at age 11. The correlation between PbB at 11 and 21 years of age was weak but significant (r = 0.19; $p<0.001$). The PbB for the two complete samples at ages 11 and 21 fell by 56%. The PbB in the same group of individuals fell by 53% over the decade (geometric mean 10.2 vs. 4.8 µg/dl; $p<0.001$) and the decline for females (9.8–3.4 µg/dl; 65%) was much greater than for males (10.5–6.0 µg/dl; 43%).

The values of PbB for the various categories of exposure risk are shown in Table 2. There are clear dose–response relationships for cigarette smoking and alcohol consumption. The results of multiple regression analysis of log transformed PbB on the various categories of exposure risk are shown in Table 3. PbB was found to be significantly associated with male gender, high risk occupational activities, lead-related recreational exposure, cigarette smoking, and living close to a main road. The associations with SES and alcohol consumption were not significant.

Because the analysis is based on log-transformed PbB, the exponents of the regression coefficients (β) in Table 3 can be interpreted as indicators of the increase in PbB associated with a particular risk factor. Thus, the exponent of the intercept is a measure of the mean PbB for females living more than 50 meters from a main road who do not smoke and are not exposed to lead through either occupational or recreational exposure. The exponent for male gender indicates that PbB for males is 1.65 times that for females and, similarly, for those involved in high risk occupations, the PbB is 1.31 times those that are not. The multiple regression coefficient of 0.54 accounts for 29% of the variance in PbB.

Respiratory protection was used by 41% of those involved in high risk occupations. The lead level in those using protection (geometric mean 7.0 µg/dl; n = 41) was not significantly different from those not using protection (geometric mean 6.6 µg/dl; n = 96).

Discussion

As pointed out in previous lead-related studies of the DMHDS (*10,11*), the sample, drawn from one urban area of New Zealand, is socioeconomically advantaged and underrepresentative of Maori and other Polynesians. The results at 21 years of age are similar to those at age 11 in that the distribution is log normal and the geometric mean for males is higher than that for females. The decline in PbB for the total sample over the 10-year period (56%) was in close agreement with the decline in the group whose blood was analyzed at both times (53%). The decline for females (65%) was less than that for males (43%).

The PbB in the DMHDS cohort can be compared with values measured since 1974 in groups of adults residing in Christchurch,

the other main urban centre in the South Island of New Zealand (*20,21*). The groups include males and females over 17 years of age representative of the Christchurch population with no known exposure to lead. The arithmetic mean PbB for 1993–1994 in Christchurch was 5.8 (SD = 2.4) µg/dl for males (n = 66) and 3.9 (SD = 1.7) µg/dl for females (n = 80) compared with geometric means in the DMHDS of 6.0 (95% CI, 5.7–6.4) µg/dl and 3.2 (95% CI, 3.1–3.4) µg/dl for males and females, respectively. The similarity in values suggests the Dunedin sample of 21-year-old adults is representative of adult New Zealanders.

Table 1. Comparison of numbers exposed to risk factors for lead between the Dunedin Multidisciplinary Health and Development Study sample giving blood at 21 years of age (n = 779) and those seen but not consenting to venipuncture (n = 170)

Risk factor	No. sampled (%)	Not sampled (%)	χ^2 (df)	p-value
Males	411 (52.8)	73 (42.9)	5.38 (1)	0.02
High risk occupation	137 (17.6)	31 (18.2)	0.40 (1)	0.84
Recreational exposure	77 (9.9)	15 (8.8)	0.17 (1)	0.67
Residence near a main road	467 (60.3)	99 (60.4)	0.00 (1)	0.98
Daily smokers	253 (32.5)	77 (45.6)	10.48 (1)	0.001
Frequency of alcohol use				
Never	105 (13.5)	27 (16.2)		
<15 times/month	593 (76.2)	124 (74.3)	0.84 (2)[a]	0.66[a]
≥15 times/month	80 (10.3)	16 (9.6)		

[a]χ^2 and p-value for comparison of the three categories of alcohol use.

Table 2. Blood lead levels in the Dunedin Multidisciplinary Health and Development Study at 21 years of age in relation to gender, socioeconomic status (SES), and exposure to lead via residential, occupational, and recreational factors as assessed by questionnaire (n = 779)

Risk factor	Categories	Number	Geometric mean PbB (µg/dl)	95% CI (µg/dl)
Gender	Males	411	6.0	5.7–6.4
	Females	368	3.2	3.1–3.4
SES of parents at age 15	High SES	395	4.3	4.0–4.6
	Low SES	384	4.7	4.4–5.0
Residence[a]	<50m from main road	467	4.7	4.5–5.0
	>50m from main road	308	4.1	3.8–4.5
Occupation	High risk	137	6.0	5.4–6.7
	Low risk	642	4.1	3.9–4.3
Recreation	High risk	77	7.6	6.8–8.5
	Low risk	702	4.2	4.0–4.4
Smoking[a]	Nonsmoking	526	4.2	3.9–4.4
	<10 cigarettes/day	83	4.8	4.2–5.5
	≥10 cigarettes/day	169	5.4	5.0–6.0
Alcohol use[a]	Abstainers	105	3.9	3.4–4.4
	<15 occasions/month	593	4.5	4.2–4.7
	≥15 occasions/month	80	5.5	4.9–6.2

[a]One or more missing values

Table 3. Multiple regression analysis of the association of blood lead level and various risk factors for lead in the Dunedin Multidisciplinary Health and Development Study giving blood at 21 years of age

Independent variable	Regression coefficient β	Standard error	Exponent[a]
Intercept	1.00	0.04	2.72
Male gender	0.50	0.04	1.65
High risk occupation	0.27	0.06	1.31
Residence near a main road	0.15	0.04	1.16
Smoking <10 cigarettes/day	0.19	0.07	1.21
Smoking ≥10 cigarettes/day	0.23	0.05	1.26
Recreational exposure	0.26	0.07	1.30

[a]See text for explanation.

The PbB in Christchurch adults declined dramatically between 1981 and 1985, followed by a slower but steady decline thereafter. The values for males were consistently higher than for females, but the two follow an approximately parallel course. The PbB in Christchurch adults fell by 49% between 1983–1984 and 1993–1994, in agreement with the 53% fall in the DMHDS despite the difference in age. While the decrease in the DMHDS between 11 and 21 years may result from a change in lead metabolism with age, it is likely some is due to reduced environmental exposure. The weak correlation between PbB at ages 11 and 21 is not surprising, given that it mainly reflects current lead status and recent exposure (22,23).

The decline in PbB in the United States has been studied by the National Health and Nutrition Examination Surveys (NHANES) in 1976–1980 (NHANES II; n = 9832) and 1988–1992 (NHANES III phase 1; n = 12,119) (14). The geometric mean PbB for persons 1 to 74 years of age dropped 78% from 12.8 (95% CI, 12.4–13.3) µg/dl to 2.8(95% CI, 2.7–3.0) µg/dl, probably due to the removal of lead from gasoline and soldered cans. The decline was similar across age groups, sex, urban status, and income levels. Comparison with the drop in the DMHDS suggests PbB has declined significantly faster in the United States than in New Zealand over a similar time period. Given that both countries reduced lead in soldered cans and paint throughout the 1980s, the slower decline in New Zealand probably reflects the difference in use of leaded gasoline during the sample period. In the United States, leaded gasoline was phased out over the decade 1976–1986, whereas in New Zealand, a similar phase out only commenced in 1986 and was not complete until early 1996.

The association between PbB and living within 50 meters of a busy road is not surprising, given that vehicle exhaust from cars using leaded gasoline is a major contributor to lead uptake (24). Place of residence in relation to a main road has been previously shown to be a major determinant of PbB in children (25). The association of PbB with high risk occupations is well established, but the association with recreational use has been previously confined to lead exposure in recreational shooters (26). The fact that the sample of the DMHDS who gave blood contained proportionally fewer smokers and had higher academic achievement than those who did not suggests that our results may slightly underestimate the PbB in the DMHDS as a whole.

With the elimination of lead from gasoline in New Zealand in early 1996, occupational and recreational activities, together with the legacy of lead-based paint, are likely to remain significant sources of lead exposure. Both ingestion and inhalation are important routes of exposure to lead so that education of workers regarding personal hygiene and the use of respiratory protection is necessary. The relatively low use of respiratory protection by those of the DMHDS cohort involved in high risk occupations suggests considerable improvement is required in this area. From a public health point of view, environmental and biological monitoring remain important.

REFERENCES

1. Alperstein G, Reznik RB, Duggin GG. Lead: subtle forms and new modes of poisoning. Med J Aust 155:407–409 (1991).
2. Ziegler EE, Edwards BB, Jensen RL, Mahaffey KR, Fomon SJ. Absorption and retention of lead by infants. Pediatr Res 12:29–34 (1978).
3. Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood. An 11 year follow-up report. New Engl J Med 322:83–88 (1990).
4. Ferguson DM, Horwood LJ, Lynskey MT. Early dentine lead levels and subsequent cognitive and behavioural development. J Child Psychol Psychiatry 34:215–227 (1993).
5. Haenninen H, Hernberg S, Mantere P, Vesanto R, Jalkanen M. Psychological performance of subjects with low exposure to lead. J Occup Med 20:683–689 (1978).
6. Stollery BT, Broadbent DE, Banks HA, Lee WR. Short term prospective study of cognitive functioning in lead workers. Br J Ind Med 48:739–749 (1991).
7. Schwartz J. Lead, blood pressure and cardiovascular disease in men. Arch Environ Health 50:31–37 (1995).
8. Rosen JF. Adverse health effects of lead at low exposure levels: trends in the management of childhood lead poisoning. Toxicology 97:11–17 (1995).
9. Pocock SJ, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. BMJ 309:1189–1197 (1994).
10. Silva PA, Hughes P, Faed JM. Blood lead levels in Dunedin 11-year-old children. NZ Med J 99:179–183 (1986).
11. Silva PA, Hughes P, Williams S, Faed JM.

12. Glass B, Hodgkinson E. Guidelines for the medical surveillance of lead workers. Wellington, NZ:Occupational Safety and Health Service, Department of Labour, 1994.
13. Nunez CM, Klitzman S, Goodman A. Lead exposure among automobile radiator repair workers and their children in New York City. Am J Ind Med 23:763–777 (1993).
14. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Flegal KM, Matte TD. The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). JAMA 272:284–291 (1994).
15. Royal Society of New Zealand. Lead in the environment in New Zealand. Miscellaneous Series No 14. Wellington:Royal Society of New Zealand, 1986.
16. Silva PA. The Dunedin Multidisciplinary Health and Development Study: a 15 year longitudinal study. Paediatra Perinat Epidemiol 4:76–107 (1990).
17. Walmsley TA, Sise JA, Hinton D. Blood lead levels—population base data. In: Proceedings of the NZ Trace Elements Group Conference, Lincoln College, Canterbury, 1988. Canterbury:NZ Trace Element Group, 1989;125-131.
18. Hinton D, Cresswell BCL, Janus ED, Malpress WA. Industrial lead exposure: a review of blood levels in South Island industries, 1974–1983. NZ Med J 97:769–773 (1984).
19. Elley WR, Irving JC. Revised socioeconomic index for New Zealand. NZ J Educ Stud 11:26–36 (1976).
20. Walmsley TA, Grant S, George P. Trends in adult blood lead levels in New Zealand, 1974–1994. The New Zealand Public Health Report, Vol 2 (No 10). Wellington, NZ:Public Health Commission, 1995;81–82.
21. Hinton D, Coope PA, Malpress WA, Janus ED. Trends in blood lead levels in Christchurch (NZ) and environs 1978–1985. J Epidemiol Community Health 40:244–248 (1986).
22. Rabinowitz MB, Wetherill GW, Kopple JD. Kinetic analysis of lead metabolism in healthy humans. J Clin Invest 58:260–270 (1976).
23. Lyngbye T, Jorgensen PJ, Grandjean P, Hansen ON. Validity and interpretation of blood lead levels: a study of Danish school children. Scand J Clin Lab Invest 50:441–449 (1990).
24. Menkes DB, Horrocks JB. New Zealand petrol—time to get the lead out? NZ Med J 107:97–99 (1994).
25. Romieu I, Palazuelos E, Meneses F, Hernandez-Avila M. Vehicular traffic as a determinant of blood-lead levels in children: a pilot study in Mexico City. Arch Environ Health 47:246–249 (1992).
26. George PM, Walmsley TA, Currie D, Wells JE. Lead exposure during recreational use of small bore rifle ranges. NZ Med J 106:422–424 (1993).

Blood lead intelligence, reading attainment and behaviour in eleven-year-old children in Dunedin, New Zealand. J Child Psychol Psychiatry 29:43–52 (1988).